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(FILE 'HOME' ENTERED AT 07:35:50 ON 05 NOV 2003)

FILE 'MEDLINE' ENTERED AT 07:35:56 ON 05 NOV 2003

L1	12 S REVIEW AND HEME(15W) BIND?
L2	339 S REDOX(25W) HEME
L3	4 S L2 AND REVIEW
L4	888 S REDOX(10W) PROTEIN
L5	23 S L4 AND REVIEW
L6	3 S L5 AND HEME
L7	35 S L4 AND HEME(15W) PROTEIN

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L7 ANSWER 16 OF 35 MEDLINE on STN
 AN 1999371536 MEDLINE
 DN 99371536 PubMed ID: 10443936
 TI Expression, purification, and biochemical characterization of SAG, a ring finger **redox**-sensitive **protein**.
 AU Swaroop M; Bian J; Aviram M; Duan H; Bisgaier C L; Loo J A; Sun Y
 CS Department of Molecular Biology, Parke-Davis Pharmaceutical Research, Division of Warner-Lambert Company, Ann Arbor, MI 48105, USA.
 SO FREE RADICAL BIOLOGY AND MEDICINE, (1999 Jul) 27 (1-2) 193-202.
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 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199911
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 AB We recently reported the cloning and characterization of SAG (sensitive to apoptosis gene), a novel zinc RING finger protein, that is redox responsive and protects mammalian cells from apoptosis. Here we report the expression, purification, and biochemical characterization of SAG. Bacterially expressed SAG is brown in color and dithiothreitol (DTT)-sensitive. SAG forms large oligomers without DTT that can be reduced into a monomer in the presence of DTT. These features help us to purify SAG using the chromatography with or without DTT. Likewise, purified SAG is redox sensitive. Upon H2O2 exposure, SAG forms oligomers as well as monomer doublets due to the formation of the inter- or intramolecular disulfide bonds, respectively. This process can be reversed by DTT or prevented by pretreatment with the alkylating reagent, N-ethylmaleimide (NEM). Although SAG contains two putative **heme**-binding sites and a RING finger domain, the **protein** appears not to bind with heme and to lack transcription factor activity as determined in a Gal4-fusion/transactivation assay. Wildtype, but not RING finger domain-disrupted SAG mutants, prevents copper-induced lipid peroxidation. These results, along with our previous observations, suggest that SAG is an intracellular antioxidant molecule that may act as a redox sensor to buffer oxidative-stress induced damage.

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